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WESTERN LARCH NEEDLE BLIGHT AND NEEDLECAST

in the NORTHERN REGION

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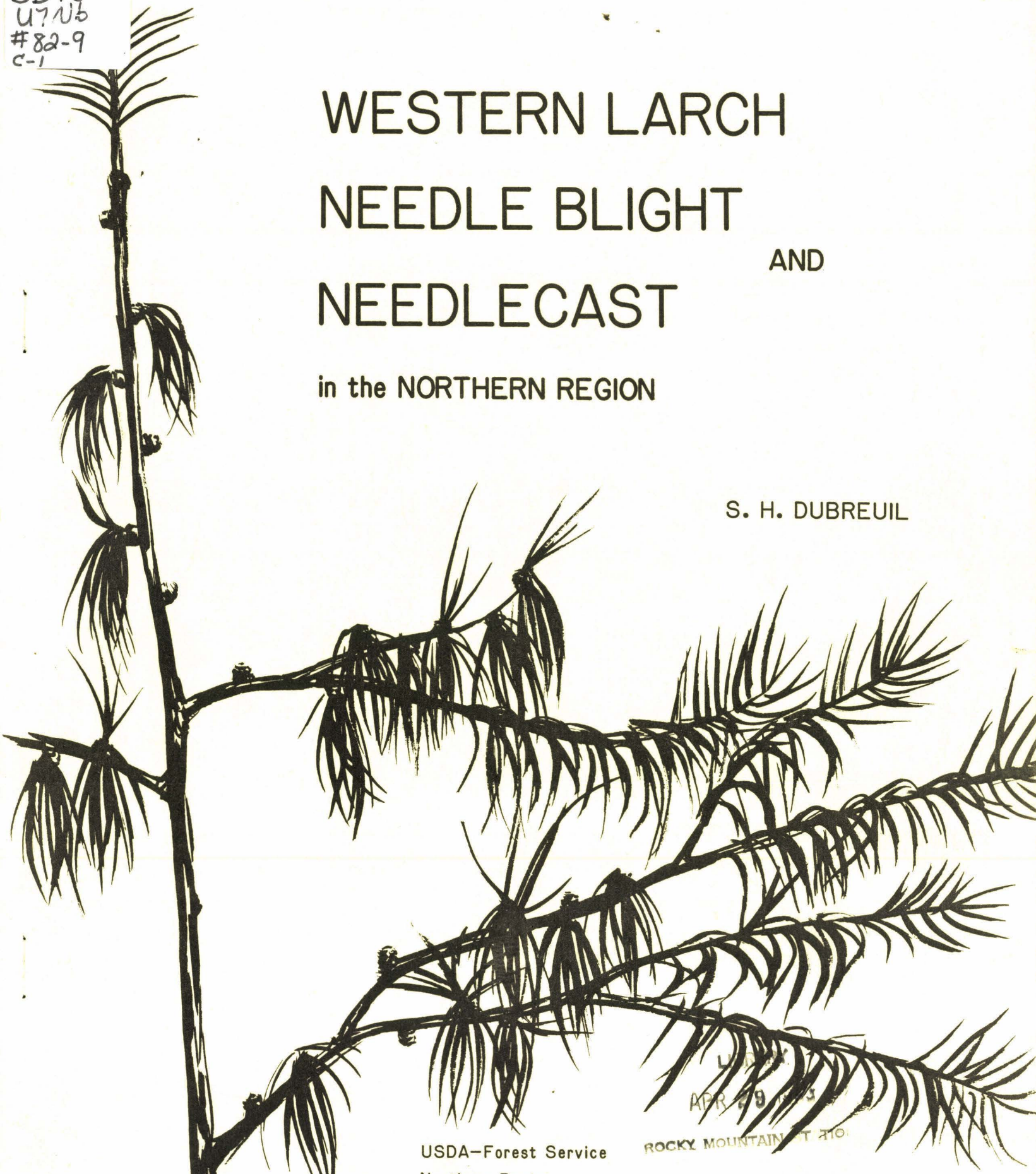

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WESTERN LARCH NEEDLE BLIGHT AND NEEDLECAST IN THE NORTHERN REGION

by

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ABSTRACT

Epidemics of western larch (Larix) occidentalis Nutt.) needle diseases caused by Hypodermella laricis Tub. and Meria laricis Vuill. occur periodically in the Northern Region. Damage from Hypodermella blight appears in mid-May to mid-June. Blighted leaves remain attached to spur shoots. Rainfall during leaf expansion favors spore production and infection. Meria needlecast increases defoliation throughout summer as long as periodic rainfall occurs. Leaves are shed 2 to 4 weeks after infection.

Damage includes (1) radial growth loss equal to the amount of defoliation, (2) death of some seedlings after 2 or 3 successive years complete defoliation, (3) seedling height growth reduction from terminal shoot death, (4) root mortality which can predispose trees to root pathogens.

Bordeaux mixture (PBI/Gordon Corp.) can be used to reduce damage in nurseries and ornamentals.

INTRODUCTION

Western larch (Larix occidentalis Nutt.) needle diseases were severe in the Northern Region during spring and summer of 1980 and 1981. Defoliation varied from slight in some trees to complete in others and seedlings were severely affected. Although disease intensity varied from stand to stand, most larch stands were affected to some degree.

Two fungi were responsible for the damage: Hypodermella laricis Tub. and Meria laricis Vuill. Epidemics of one or both of these fungi have been reported peridically in northern Idaho (Ehrlich 1942; Weir 1913), western Montana (Cohen 1967), and southern British Columbia (Vanderal 1970); the natural range of western larch. High infection rates by Hypodermella are associated with rainfall during bud burst and early leaf development. Meria is favored by frequent rainfall from early leaf development continuing throughout the summer.

Growth loss and mortality resulting from epidemics of these fungi on western larch have not been studied. Effects from artificial and insect defoliation of western larch and other species of Larix were consistent regardless of cause (discussed later in IMPACTS section). This indicates that the effects of defoliation caused by Hypodermella are probably similar.

Larch needle disease epidemics of 1980 and 1981 are discussed from a historical perspective. Life histories of causal fungi, a brief review of literature regarding impacts of larch defoliation, and control treatments are included.

LIFE HISTORY

Hypodermella laricis Tub.: larch needle blight

An Ascomycete of the order Phacidiales, H. laricis, produces elongate black fruiting bodies on the undersides of leaves (fig. 1).

Figure 1.--Western larch needle blight (A) and needlecast (B).

A. Red-brown needles attached to spur; elongate black fruiting bodies of Hypodermella laricis.

B. Discoloration pattern of Meria laricis-infected leaves.

The fruiting body, a hysterothecium, contains spores in sacs called asci (fig. 2). In western Montana, the spores are mature in February (Cohen 1967) (fig. 3). During spring rain, usually in mid-April or early May, the walls of the hysterothecium absorb water and split the fruiting body open length-

wise exposing the sacs containing the spores. The sacs burst open and the spores are released. If the larch buds have begun to open and the leaves are exposed, the spores which land on leaves germinate and penetrate the surface. Once leaves have reached maturity they are much more resistant to infection. If rainfall does not coincide with early leaf growth, few of the spores will successfully infect leaves and the disease will be light that year.

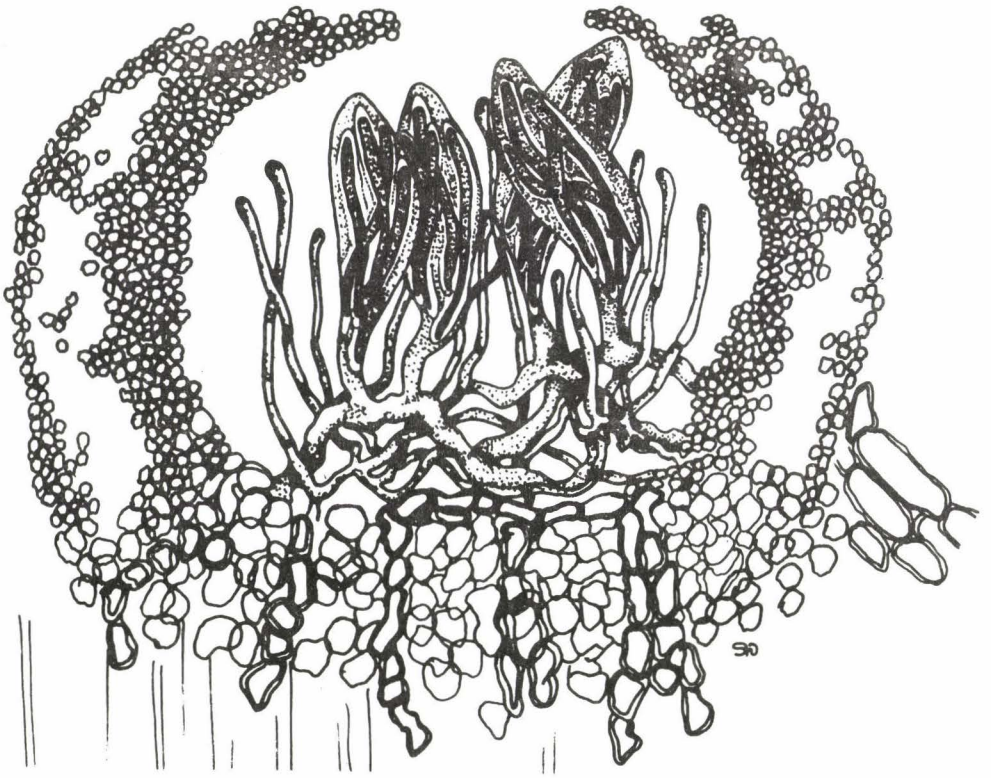

Figure 2.--Fruiting body of Hypodermella laricis. Thread-like spores within sacs are produced inside the cavity of the black hysterothecium on the undersides of leaves.

Approximately 1 month following infection, usually mid-May to mid-June, the needles turn brown and droop, remaining attached to the spur shoots (Cohen 1967). The process of leaf death may take only 3 days, giving the appearance that entire stands of larch have suddenly died.

Western larch has long shoots and short (or spur) shoots. Spur shoots bear the majority of the foliage. Long shoots are responsible for height growth and branch extension. Spur shoots are not invaded by Hypodermella (Cohen 1967), but it is uncertain whether long shoots become infected.

In western Montana, during 1956 and 1957, Cohen (1967) reported that infection in leaves of long shoots was light compared to infection in leaves of short shoots (spur shoots). This low infection was probably due to delayed

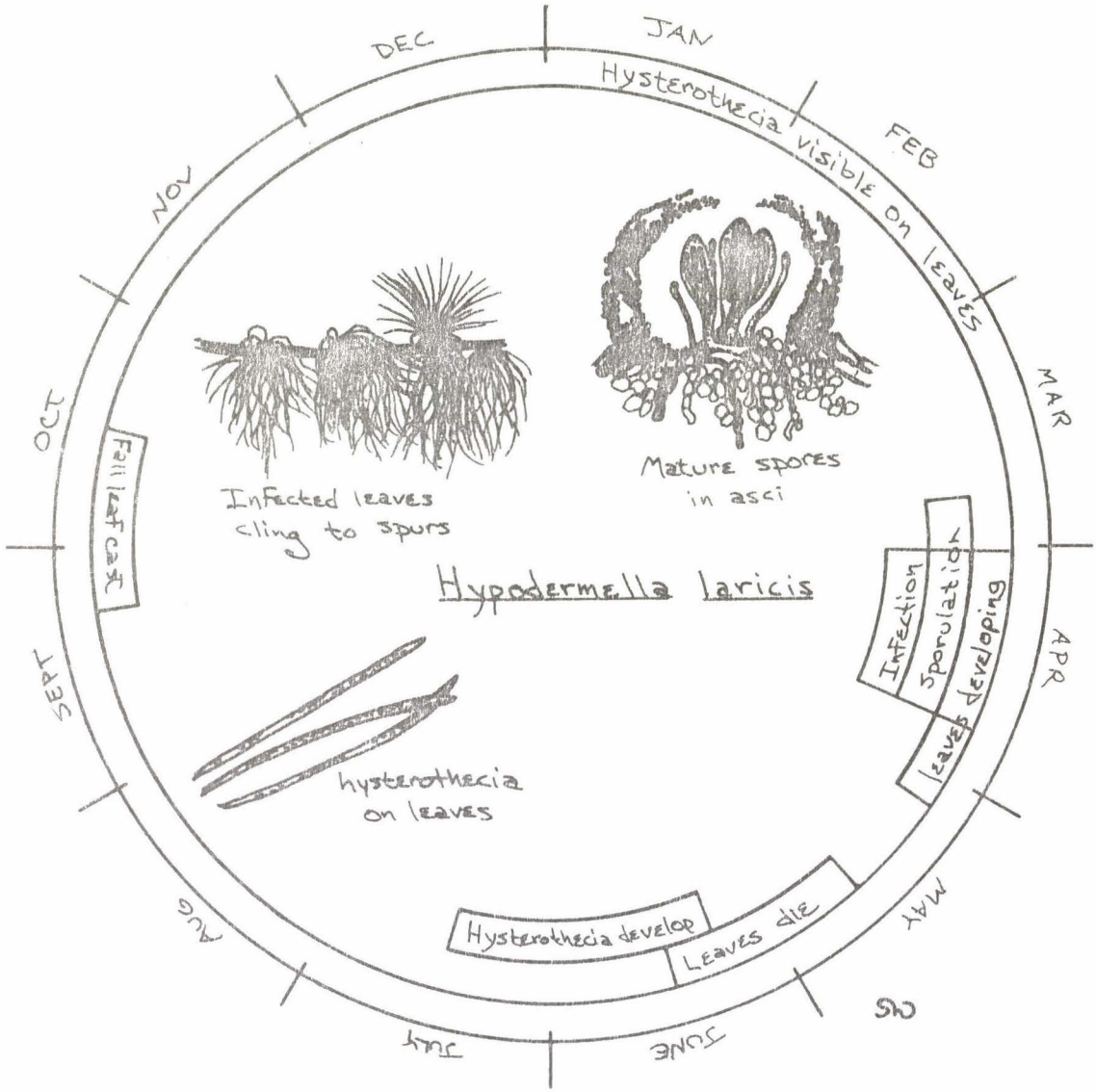

Figure 3.--Life cycle of Hypodermella laricis causing needle blight of western larch.

bud burst of long shoots. The supply of spores would have been largely spent by the time leaves of long shoots had been produced. Cohen observed no death of long shoots resulting from Hypodermella infections, although Peace (1962) and Weir (1913) found this a consequence of the disease. Recent observations (July 1981) in the Red Ives District of the Idaho Panhandle National Forests of northern Idaho indicate that H. laricis invades or otherwise causes the death of long shoots. In 50 randomly selected young trees (<1 cm diameter at 0.3 m height), 81 percent of the long shoots were dead in July following a severe infection by both Hypodermella and Meria. Meria has not been reported to kill long shoots. Late frost can cause symptoms similar to those observed in the long shoots of needlecast infected trees; however, no other tree species in the stands were showing symptoms of frost damage to new growth so it is unlikely that this was the source of damage in the larch.

Elongate, black fruiting bodies appear on the undersides of leaves in July or August. Spores within them are still immature in October. By January, however, the spores have matured and are ready for above-freezing temperatures and rain in the spring (Cohen 1967).

When buds resume growth in the spring, differences become apparent between spur shoots which bore infected leaves the previous year and those which did not. Buds which had borne infected leaves open later than buds which had healthy leaves the previous year. The leaves produced on the shoots which had infected leaves the previous year remain dwarfed to half or less the length of normal leaves and there are fewer leaves per shoot (Cohen 1967). The dead leaves may remain attached to spur shoots for 1 or 2 years (Cohen 1967).

Meria laricis: Vuill. larch needlecast

Meria laricis was first reported in the Northern Region in 1942 (Ehrlich 1942). The disease was probably not a new arrival but had been previously overlooked due to its close occurrence with Hypodermella, a more readily recognizable fungus. Meria is a Hyphomycete of the order Moniliales. Spores are produced on simple fruiting structures called conidiophores which protrude in groups through stomatal openings (fig. 4). Conidiophores can be seen by using a hand lens and appear as rows of small white dots on the undersides of leaves. While rain aids in the dispersal and germination of spores, it is apparently not a requirement for spore production or release (Peace 1962).

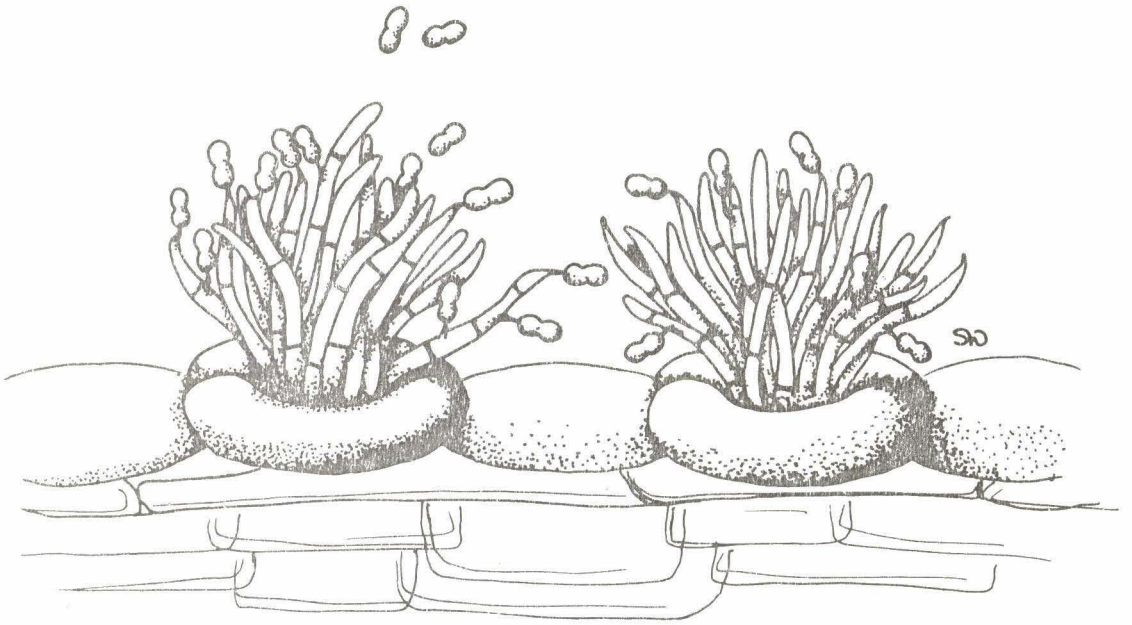

Figure 4.--Spore production of Meria laricis. Peanut-shaped spores produced on conidiophores which emerge through stomata on the undersides of leaves.

Sources of inoculum in the spring are infected leaves on the ground and tufts of infected needles on the terminal shoots of seedlings. Spores are carried by wind or splashing rain to the newly emerged leaves. Infection generally occurs at the tip or midportions of developing leaves, probably because young leaf tissue is more resistant to infection (Peace 1933).

After infection, the fungus grows toward the base of the leaf. In early May, yellowing and withering of infected needle tips is noticeable (fig. 1). Leaves eventually turn brown and are cast. Early in the spring most symptoms on long shoots will be in leaves near the base of shoots because it takes some time for symptoms to develop (Peace 1933).

Spores are produced on newly cast leaves and reinfect previously uninfected leaves. The cycle from infection to sporulation (spore production) requires approximately 2 to 4 weeks (fig. 5). If favorable moisture and temperature conditions prevail throughout the summer, the inoculum load of Meria continues to increase and resulting damage excalates.

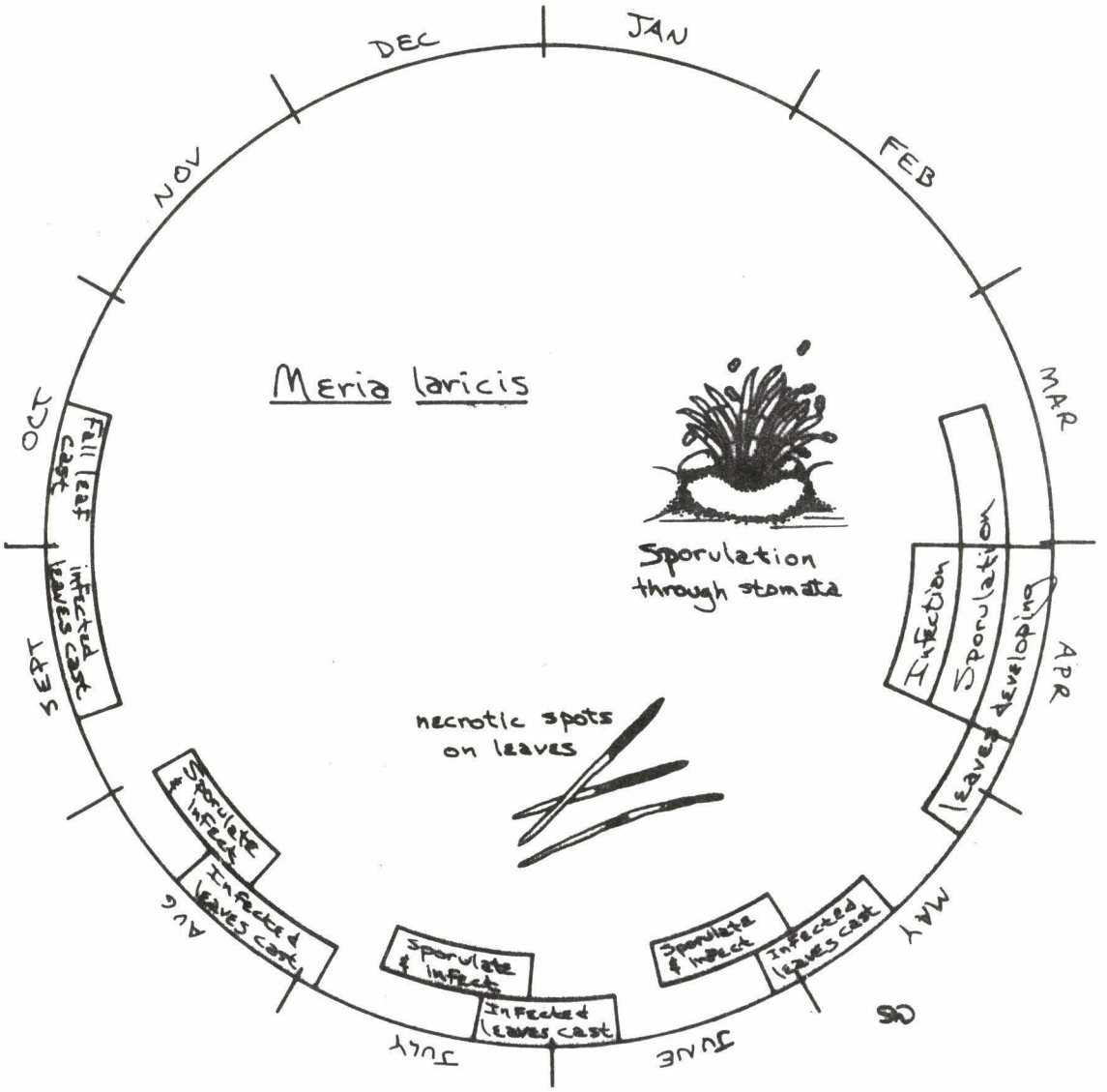

Figure 5.--Life cycle of Meria laricis causing needle cast of western larch.

IMPACTS

Epidemics of Hypodermella or Meria in the Northern Region have been reported about every 10 years. The first report available (Weir 1913) indicates that during 1912 and 1913 severe larch needle disease occurred. Schmitz (1923) reported extremely severe defoliation in 1922 as a culmination of about 5 years of heavy infections. Ehrlich (1942) reported severe infections in 1941 and 1942 and Cohen (1967) reported that 1956 was a heavy infection year. Annual conditions reports from the Region refer to heavy infections in 1964 (Tunnock and Toko 1965) and 1965 (Tunnock et al. 1966). A 1978 epidemic in northern Idaho was reported to have been primarily Meria (Robinson et al. 1979).

Infection was notably light in 1979, but in 1980 and again in 1981, infections by larch needle blight and needlecast were severe (Kohler et al. 1981). In the spring of 1980 an aerial survey for larch casebearer was undertaken by Northern Region Forest Pest Management but was abandoned because the prevalence of lach needle disease made detection of casebearer damage impossible. However, the most heavily discolored stands of larch were delineated on the aerial survey maps for all the Region [1/] with the exception of the Idaho Panhandle National Forests (IPNF) (fig. 6). Damage in the IPNF was in essentially all larch stands. General distribution of larch in the IPNF was superimposed on the map as the likely distribution of larch leaf diseases in 1981 (fig. 6). This was substantiated by on-the-ground observations of pathologists and by foliar samples and reports received from Forest personnel. The heaviest infections in the Region in 1980 and 1981 were reported in the IPNF's, especially the St. Joe Forest.

Severe Hypodermella or Meria infection can kill the whole tree, kill portions of the tree, or reduce growth. Spur shoots with 2 years' successive total or near total infection by Hypodermella died (Cohen 1967, Schmitz 1923). Branches with a high rate of spur mortality resulting from Hypodermella infections also frequently die (Schmitz 1923). All roots died following 3 successive years of complete defoliation in early spring by artificial means (Graham 1931). Graham (1931) reported amounts of root death from 4 successive years of 75 percent defoliation to be insignificant; however, Ives and Nairn (1966) found the amounts of root death after 4 years of 70 percent defoliation to be significant. The role of root killing by defoliation in predisposing roots to invasion by pathogens or mining insects should be evaluated. This may prove a more important source of loss than the direct effects of defoliation.

Long shoot mortality with resulting loss in height growth has been reported by Peace (1962) and by Weir (1913). On July 21, 1981, I sampled 50 larch seedlings (<1 cm d.b.h.) on the Red Ives District (IPNF) for long shoot mortality. All of the seedlings had 90 to 100 percent defoliation by Hypodermella and Meria. Of 802 long shoots tallied, 622 had been killed. Individual trees averaged 81 percent mortality of long shoots. Dead long shoots were brown and curved with some of the foliage, also brown and shriveled, clinging to them. Only about 25 percent of the trees had dead terminals. In saplings and larger trees, infections by both fungi were

1/ Aerial survey was performed by Scott Tunnock, Forest Pest Management, Northern Region.

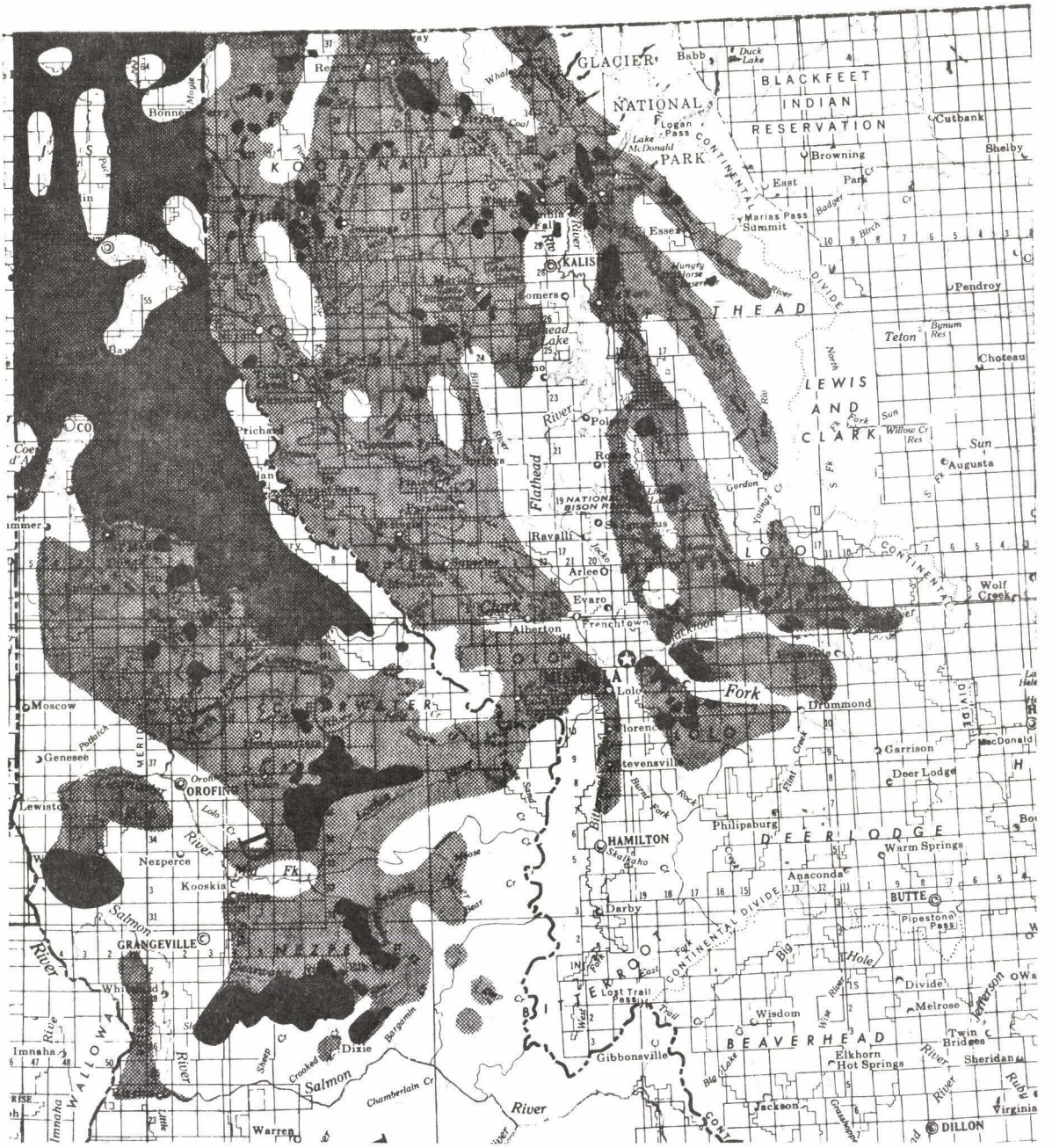

Figure 6

Larch defoliation

Distribution of western larch in the Northern Region. Most of this area had Hypodermella blight and Meria needle cast of larch in 1981.

Extreme defoliation levels detected in 1980 aerial survey.

Western larch not surveyed in 1980. Most of this area had very heavy infections of Hypodermella blight and Meria needle cast in 1981.

concentrated in the lower 2/3 to 3/4 of the crowns. The 50 trees sampled were marked in July and 46 of these were relocated on October 3, 1981, and examined to determine if they had survived the infections. Thirteen of the 46 seedlings (28 percent) had died. They had no live stem cambium and most of the roots were dead. Ehrlich (1942) reported that heavy infection by both Hypodermella and Meria in northern Idaho in 1941 and 1942 had killed "a considerable amount" of reproduction. Leaphart and Denton (1961) reported tree killing by Hypodermella to be rare but that small seedlings were occasionally killed. In small European larch (Larix decidua Mill.) nursery stock infected with Meria, a few of the weakest plants died (Peace 1962). Graham (1931) reported 100 percent defoliation for 3 successive years killed eight out of ten, 5-year-old artificially defoliated tamarack.

Growth loss resulting from severe infections is probably the greatest effect although there is little data demonstrating that infections of either of these two fungi can cause growth loss. Peace (1962) stated that small European larch nursery stock growth loss caused by Meria resulted in a higher percentage of cull plants.

Spur shoots are killed after 2 successive years of infection by Hypodermella (Cohen 1967) and are weakened as evidenced by late bud burst (Cohen 1967, Weir 1913), fewer leaves/spur (Cohen 1967), and stunting (Cohen 1967, Weir 1913) and chlorosis (Cohen 1967) of leaves after 1 year infection.

Studies on artificial defoliation and insect defoliation of Larix spp. have yielded consistent results regarding growth loss. Generally, the rate of radial growth loss equals the rate of defoliation (Graham 1931, Kulman 1971). Thus, a 70 percent defoliation results in approximately 70 percent radial growth loss. Graham (1931) found considerable variation between individual tamarack (Larix laricina DuRoi) in their initial response to complete artificial defoliation. A few trees actually increased growth the year they were defoliated but declined rapidly afterward. First year increases in growth were largely due to a second flush of foliage which was produced with a corresponding period of radial growth (Graham 1931, Mott et al. 1957). Trees not entirely defoliated did not show an increase in growth and responded immediately by decreasing their growth to match amount of foliage lost.

Tamarack spurs which had been artificially defoliated responded in much the same manner as spurs defoliated by Hypodermella. In the year following artificial defoliation, spurs produced smaller and fewer leaves than nondefoliated spurs (Graham 1931). Spurs died after 2 to 3 years of artificial defoliation. When most of the spurs on a branch were dead, the branch died. Hypodermella and Meria most often occur together on western larch in the northern Region. Combined effects of these fungi may have considerably greater impact than the effects of either fungus alone. Although Hypodermella is more effective at killing trees, it causes damage only during a relatively limited portion of the growing season. On the other hand, Meria is capable of almost continuous reinfection of foliage throughout the spring and summer, thus reducing the chances of leaves escaping infection, and causing continuous reduction in photosynthetic area. Larix spp. respond to excessive artificial and insect defoliation by producing a second flush of foliage in the same season (Graham 1931). If this

behavior is also a consequence of excessive defoliation by Hypodermella or Meria, as suggested by Weir (1913), then the ability of Meria to infect leaves of second flush growth could further drain resources of the tree.

Effects of larch defoliation are summarized as follows:

1. Radial growth loss equals the amount of defoliation.

2. A temporary growth increase may result from complete defoliation.

3. Seedlings may be killed after 2 or 3 successive years of complete defoliation.

4. Significant root mortality in seedlings and saplings may result from 4 or more successive years of 70 percent defoliation.

5. Terminal long shoot death results in direct loss of height growth.

CONTROL

Until the actual impact of needle disease of larch has been adequately investigated both in nurseries and in forests, desirability of controlling them will be uncertain. There are currently no chemical control measures practicable under forest conditions. However, nursery and ornamental larch can be protected with fungicidal sprays. One percent lime-sulphur has been used in British nurseries for controlling Meria (Peace 1936). Bordeaux mixture (copper hydroxide) PBI/Gordon Corporation (EPA 33955-97) is registered for control of needlecasts in Idaho and Montana. Follow all label instructions carefully.

Suggested spray schedule for Hypodermella laricis:

1. When leaves are just emerging.
2. When the leaves are about half grown.

Once the leaves have reached maturity, they are apparently too resistant to infection to incur significant damage from Hypodermella.

Suggested spray schedule for Meria laricis (Peace 1936):

1. First spray in early March when buds begin to expand and the green tips of leaves become visible.

2. Spray again in early April when growth resumes.

3. Reapply at 2- to 3-week intervals until dry weather sets in or until end of July.

Most tree growth should be complete by August and the effects of infection at that point would be minimal.

Although the efficacy of the system is as yet untested, Cohen (1967) has presented strong evidence that epidemics of Hypodermella can be predicted. High rates of infection correspond to rainfall during the period from leaf emergence to near maturity of leaves. Disease forecasting based on weather conditions has long been used to prevent damage to field crops (Bourke 1970). Similar forecasting might be useful in alleviating the threat from larch needle blight in nursery and young ornamental larch.

Considerable variation in susceptibility to both species of fungi in our native populations of western larch is suspected. A proportion of larch in every stand examined had notably less infection by either or both fungi. High levels of infection of other larch in the stands and the lack of distinction in the position of the less-affected individuals made escape from infection an unlikely explanation for this phenomenon. Some individuals were severely infected with Meria alone while the remainder of the stand was heavily infected by both fungi. Others were only infected by Hypodermella to a measurable degree, and still others were relatively free of infection by either species, appearing strikingly green when compared to their neighbors.

Apparently some degree of damage control may be attainable using selective thinning or seed tree harvests favoring least-infected individuals. However, treatment could be done only in years when infection is heavy enough to be easily detected.

Perhaps selective breeding of larch for resistance to needle blight and needlecast should be included in a western larch tree improvement program.

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